

Amendments to the Claims:

This listing of claims will replace all prior versions, and listings, of claims in the application:

Listing of Claims:

1. (Original) A method for treating cancer comprising administering to a subject in need of such treatment a therapeutically effective amount of
 - (a) a member selected from an inhibitor of inosine monophosphate dehydrogenase (IMPDH), an enantiomer of such a compound, a prodrug of such a compound, a pharmaceutically acceptable salt of such a compound, and combinations thereof; and
 - (b) an agent that inhibits a cellular process regulated by GTP or ATP.
2. (Original) The method of claim 1, wherein the agent that inhibits a cellular process regulated by GTP is selected from the group consisting of an inhibitor of α -tubulin polymerization, a prodrug therefor, a pharmaceutically acceptable salt thereof, and combinations thereof.
3. (Original) The method of claim 2, wherein the IMPDH inhibitor is selected from the group consisting of mizoribine, mizoribine aglycone, mycophenolate mofetil, tiazofurin, viramidine, and ribivarin.
4. (Original) The method of claim 2, wherein the α -tubulin polymerization inhibitor is selected from the group consisting of indanocine, indanrorine, vincristine, vinblastine, vinorelbine, combretastatin-A, and colchicine.
5. (Original) The method of claim 2, wherein the IMPDH inhibitor is mizoribine and the α -tubulin polymerization inhibitor is indanocine.
6. (Original) The method of claim 2, wherein the cancer is a slow growing cancer.

7. (Original) The method of claim 6, wherein the slow growing cancer has a high rate of α -tubulin turnover.

8. (Original) The method of claim 6, wherein the slow growing cancer is selected from the group consisting of chronic lymphocytic leukemia, chronic myelogenous leukemia, non-Hodgkins lymphoma, multiple myeloma, chronic granulocytic leukemia, cutaneous T cell lymphoma, low grade lymphomas, slow growing breast cancer, slow growing prostate cancer, and slow growing thyroid cancer.

9. (Withdrawn) A composition for treating cancer in a subject in need of such treatment comprising therapeutically effective amounts of

(a) a member selected from an inhibitor of inosine monophosphate dehydrogenase (IMPDH), an enantiomer of such a compound, a prodrug of such a compound, a pharmaceutically acceptable salt of such a compound, and combinations thereof; and

(b) an agent that inhibits a cellular process regulated by GTP or ATP.

10. (Withdrawn) The composition of claim 9, wherein the agent that inhibits a cellular process regulated by GTP is a member selected from an inhibitor of α -tubulin polymerization, a prodrug therefor, or a pharmaceutically acceptable salt thereof, and combinations thereof.

11. (Withdrawn) The composition of claim 10, wherein the IMPDH inhibitor is selected from the group consisting of mizoribine, mizoribine aglycone, mycophenolate mofetil, tiazofurin, viramidine, and ribivarin.

12. (Withdrawn) The composition of claim 10, wherein the α -tubulin polymerization inhibitor is selected from the group consisting of indanocine, vincristine, vinblastine, vinorelbine, combretastatin-A, and colchicine.

13. (Withdrawn) The composition of claim 10, wherein the IMPDH inhibitor is mizoribine and the α -tubulin polymerization inhibitor is indanocine.

14. (Original) The method of claim 1, wherein the agent that inhibits a cellular process regulated by GTP is a member selected from a precursor of 9-beta-D-arabinofuranosylguanine 5'-triphosphate (Ara-GTP), a prodrug therefore, a pharmaceutically acceptable salt thereof, and combinations thereof.

15. (Original) The method of claim 14, wherein the IMPDH inhibitor is selected from the group consisting of mizoribine, mizoribine aglycone, mycophenolate mofetil, tiazofurin, viramidine, and ribivarin.

16. (Original) The method of claim 14, wherein the precursor of Ara-GTP is selected from the group consisting of guanine arabinoside (Ara-G) and Nelarabine.

17. (Original) The method of claim 14, wherein the cancer is a lymphoma or a leukemia.

18. (Withdrawn) The composition of claim 9, wherein the agent that inhibits a cellular process regulated by GTP is a member selected from a precursor of Ara-GTP, a prodrug therefor, or a pharmaceutically acceptable salt thereof, and combinations thereof.

19. (Withdrawn) The composition of claim 18, wherein the IMPDH inhibitor is selected from the group consisting of mizoribine, mizoribine aglycone, mycophenolate mofetil, tiazofurin, viramidine, and ribivarin.

20. (Withdrawn) The composition of claim 18, wherein the precursor of Ara-GTP is selected from the group consisting of guanine arabinoside (Ara-G) and Nelarabine.

21. (Original) The method of claim 1, wherein the agent that inhibits a cellular process regulated by GTP is a member selected from an inhibitor of the de novo pathway of purine biosynthesis, a prodrug therefor, or a pharmaceutically acceptable salt thereof, and combinations thereof.

22. (Original) The method of claim 21, wherein the IMPDH inhibitor is selected from the group consisting of mizoribine, mizoribine aglycone, mycophenolate mofetil, tiazofurin, viramidine, and ribivarin.

23. (Original) The method of claim 21, wherein the IMPDH inhibitor is mizoribine.

24. (Original) The method of claim 21, wherein the IMPDH inhibitor is mizoribine aglycone.

25. (Original) The method of claim 21, wherein the inhibitor of the de novo pathway of purine biosynthesis is selected from the group consisting of L-alanosine, methotrexate, trimetrexate, 10-propargyl-5,8-dideazafolic acid (PDDF), N-[5-[N-(3,4-dihydro-2-methyl-4-oxoquinazolin-6-ylmethyl)-N-methylamino]-2-thenoyl]-L-glutamic acid (ZD1694, Tomudex), N-[4-[2-(2-amino-3,4-dihydro-4-oxo-7H-pyrrolo[2,3-d]-pyrimidin-5-yl)ethyl]-benzoyl]-L-glutamic acid (LY231514), 6-(2'-formyl-2'naphthyl-ethyl)-2-amino-4(3H)-oxoquinazoline (LL95509), (6R,S)-5,10-dideazatetrahydrofolic acid (DDATHF), 4-[2-(2-amino-4-oxo-4,6,7,8-tetrahydro-3Hpyrimidino[5,4,6][1,4]-thiazin-6yl)-(S)-ethyl]-2,5-thienoylamino-L-glutamic acid (AG2034), and N-[5-(2-[(2,6-diamino-4(3H)-oxopyrimidin-5-yl)thio]ethyl)thieno-2-yl]-L-glutamic acid (AG2009).

26. (Original) The method of claim 21, wherein the cancer comprises a population of cells deficient in the enzyme methyladenosine phosphorylase (MTAP).

27-30. (Cancelled)

31. (Withdrawn) The composition of claim 9, wherein the agent that inhibits a cellular process regulated by GTP is a member selected from an inhibitor of the de novo pathway of purine biosynthesis, a prodrug therefor, a pharmaceutically acceptable salt thereof, and combinations thereof.

32. (Withdrawn) The composition of claim 31, wherein the IMPDH inhibitor is selected from the group consisting of mizoribine, mizoribine aglycone, mycophenolate mofetil, tiazofurin, viramidine, and ribivarin.

33. (Withdrawn) The composition of claim 31, wherein the inhibitor of the de novo pathway of purine biosynthesis is selected from the group consisting of L-alanosine, methotrexate, trimetrexate, 10-propargyl-5,8-dideazafolic acid (PDDF), N-[5-[N-(3,4-dihydro-2-methyl-4-oxoquinazolin-6-ylmethyl)-N-methylamino]-2-thenoyl]-L-glutamic acid (ZD1694, Tomudex), N-[4-[2-(2-amino-3,4-dihydro-4-oxo-7H-pyrrolo[2,3-d]-pyrimidin-5-yl)ethyl]-benzoyl]-L-glutamic acid (LY231514), 6-(2'-formyl-2'naphthyl-ethyl)-2-amino-4(3H)-oxoquinazoline (LL95509), (6R,S)-5,10-dideazatetrahydrofolic acid (DDATHF), 4-[2-(2-amino-4-oxo-4,6,7,8-tetrahydro-3Hpyrimidino[5,4,6][1,4]-thiazin-6yl)-(S)-ethyl]-2,5-thienoylamino-L-glutamic acid (AG2034), and N-[5-(2-[(2,6-diamino-4(3H)-oxopyrimidin-5-yl)thio]ethyl)thieno-2-yl]-L-glutamic acid (AG2009).

34. (Withdrawn) The composition of claim 31, wherein the inhibitor of the de novo pathway of purine biosynthesis is L-alanosine.

35. (Original) The method of claim 1, wherein the agent that inhibits a cellular process regulated by GTP is an antagonist of a G-protein coupled receptor (GPCR).

36. (Original) The method of claim 35, wherein the IMPDH inhibitor is selected from the group consisting of mizoribine, mizoribine aglycone, mycophenolate mofetil, tiazofurin, viramidine, and ribivarin.

37. (Original) The method of claim 35, wherein the GPCR antagonist is selected from the group consisting of atrasentan, leuprolide, goserelin, and octreotide.

38. (Original) The method of claim 35, wherein the cancer is prostate cancer.

39. (Withdrawn) The composition of claim 9, wherein the agent that inhibits a cellular process regulated by GTP is a member selected from an antagonist of a G-protein coupled receptor (GPCR), a prodrug therefor, or a pharmaceutically acceptable salt thereof.

40. (Withdrawn) The composition of claim 39, wherein the IMPDH inhibitor is selected from the group consisting of mizoribine, mizoribine aglycone, mycophenolate mofetil, tiazofurin, viramidine, and ribivarin.

41. (Withdrawn) The composition of claim 39, wherein the GPCR antagonist is selected from the group consisting of atrasentan, leuprolide, goserelin, and octreotide.

42-50. (Cancelled)

51. (Original) A method for treating cancer comprising administering to a subject in need of such treatment a compound selected from the group consisting of mizoribine, mizoribine aglycone, prodrugs of mizoribine, and prodrugs of mizoribine aglycone, wherein the compound is administered in an amount sufficient to maintain a plasma level of the compound of between 0.5 and 50 micromolar for between 6 and 72 hours.

52. (Original) The method of claim 51, wherein the plasma level of compound is between 1 and 30 micromolar for between 8 and 48 hours.

53. (Original) The method of claim 51, wherein the plasma level of compound is between 5 and 25 micromolar for between 10 and 24 hours.

54. (Original) The method of claim 51, wherein the plasma level of compound is at least 10 micromolar for at least 12 hours.

55. (Original) The method of claim 51, wherein the compound comprises a pharmaceutically acceptable carrier.

56. (Original) The method of claim 51, wherein the compound is administered parenterally.

57. (Original) The method of claim 51, wherein the compound is administered orally.

58-62. (Cancelled)

63. (New) The method of claim 51, wherein the cancer comprises a population of cells deficient in the enzyme methyladenosine phosphorylase (MTAP).